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Some Observations on Traumatic Pericarditis in Cattle

BY

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SUMMARY.—Clinical observations are recorded on 15 cases of traumatic pericarditis and an attempt is made to correlate the symptoms with the lesions found on post-mortem *examination.*

Introduction

PERICARDITIS is one of the most frequently encountered forms of heart disease in cattle and the syndrome due to trauma from reticular penetration has been described by Arthur (1947), Formston (1949), and Blood and Hutchings (1955b).

This paper describes observations on 15 cases of bovine traumatic pericarditis received by the Veterinary School of the University of Bristol during 1954 to 1958. The object of this study was to correlate the symptoms and clinical signs with the lesions found on *post-mortem* examination. Although one animal was destroyed on admission the remaining 14 were kept from 3 to 38 days with an average of 12 days. Two animals died and the remainder were destroyed either on humanitarian grounds or because it was considered desirable to carry out a *post-mortem* examination on them at a particular stage in the disease process. A comparison was also made with a limited number of other forms of pericarditis encountered in cattle. Not all the animals were seen in the early stages whilst some were admitted as cases of reticulitis in which pericardial penetration was not at first suspected.

Observations

The Foreign Body

In the cases of traumatic pericarditis under consideration the distribution of the foreign bodies is shown in Table I. In every case the foreign body was a piece of baling wire which varied from 2 inches to 4½ inches in length, the majority being 4 inches long, usually sharp at both ends and sometimes slightly curved. A typical example is shown in Fig. 3. In two cases with severe fibrino-purulent pericarditis the foreign body was free in the reticulum

TABLE I
DISTRIBUTION OF THE FOREIGN BODIES

Total no. of cases	Cases on which rumenotomy performed	Foreign body removed at operation	Foreign body located at P.M. in		No foreign body found at P.M.
			Pericardium	Reticulum	
15	7	4	7	3	1

suggesting that pericardial penetration may have been followed by return to the reticulum. In one of these cases two similar wires were located in the duodenum penetrating the mucosa but not the serosa. This indicates that wires may pass through the stomachs and might be the explanation for the absence of a foreign body in one animal on *post-mortem* examination. The other alternatives would appear to be either regurgitation in a food bolus or gradual disintegration of the wire.

In one animal the foreign body was penetrating the pericardium and there was another track parallel to this which passed through the diaphragmatic adhesions and entered the pericardium (Fig. 2). In this case the same wire may have penetrated twice in two different tracks although it is also possible that two wires penetrated together and that one subsequently returned to the reticulum and disappeared.

Symptoms and Clinical Signs

The general symptoms shown by the animals studied were very similar to those described by Arthur (1947). When oedema was very severe the animals tended to lie down for long periods. The presence of a painful focus in the chest and anterior reticular area rendered straining painful and faeces were often passed in very small quantities with little straining effort.

The eyes often remained bright even when circulatory impairment was very severe. In this study

all except 2 of the animals were destroyed so that it is not possible adequately to describe the terminal symptoms, but it seems likely that whilst some cases may die of toxaemia others probably succumb from embarrassment of heart action by cardiac tamponade and pericardial fibrosis.

In the majority of cases appetite was not completely lost, although desire for food gradually waned, particularly as oedema became more marked. Generally ruminal movements and rumination persisted although they were reduced compared with normal animals.

Whilst the body temperature of the animals in this study was occasionally in the range 104° to 106° F., as reported by Arthur (1947) and Formston (1949), the temperature was more often 102° to 104° F. both in the early stages and throughout the course of the disease. There was frequently marked daily variation. Of 10 cases in which regular records were kept the highest temperature recorded was 105.3° F. and the maximum temperature in 5 of them was 103° F.

In all the cases studied the heart rate was over 90 per minute and in the majority it was over 100, generally being in the range 100 to 130. As cardiac embarrassment increased the pulse became weaker and terminally was thready and imperceptible.

In cases where there were no lesions in either the lungs or pleura the respiratory rate was generally in the normal range (20 to 30 per minute), but when pleural adhesions or pulmonary abscesses were present the rate was increased up to 35 to 55 per minute. The adhesions often involved the areas of lung adjacent to the pericardial sac (see *post-mortem* findings). The active pleural inflammation underlying the adhesions resulted in pain and restricted lung movement which caused a shallower respiration with a compensating increase in rate.

Venous Engorgement and Peripheral Oedema

The observations on the 15 cases of traumatic pericarditis are shown in Table II. In only 2 animals was there absence of jugular congestion characterised either by permanent cording of the vessel or the presence of a true jugular pulse. One of these was an 8-year-old bull which had shown vague symptoms for several months and the other was a cow with a history of illness 6 months before definite symptoms were shown. On *post-mortem* examination it appeared in these cases that early pericardial inflammation had been localised to an area adjacent to the site of penetration and that the adhesions had later broken down to produce a diffuse pericarditis. Neither of these animals developed oedema.

Whilst it is agreed that if all the animals had been allowed to die naturally the incidence of jugular engorgement and oedema might well have been greater than that shown in Table II it is interesting to note that in one very early case oedema was absent and there was also no peripheral oedema in an animal which had been affected for 38 days. This suggests therefore that even in long-standing cases oedema is not an invariable feature of traumatic pericarditis.

TABLE II
VISIBLE SIGNS OF CIRCULATORY DISTURBANCE

No. of cases	Jugular engorgement	Oedema			No oedema
		Brisket	Submax. space	Sternum	
15	13	11	4	1	4

In all the cases in which peripheral oedema developed (11 out of 15 animals) the brisket was involved and it was generally the primary site for the accumulation of transudate. Oedema also sometimes appeared in the submaxillary space and on the ventral aspect of the sternum.

Besides the jugulars, other superficial vessels were more pronounced, particularly the mammary veins, and the conjunctival vessels were tortuous and cyanosed. Associated with the vascular impairment, coldness of the extremities became marked and in one animal this was accompanied by frequent kicking with one hind leg suggesting that the cow might have been attempting to improve the local circulation.

E.C.G. Changes

In this study electrocardiograms were examined in 9 cases using bipolar limb leads. These records illustrated the increase in heart rate and regularity was maintained. Considering such a small number of cases no obvious deviation from normal in the character of the individual complexes could be recognised and certainly no changes similar to those described in human cases of pericarditis (Wood, 1956). The electrical axis of QRS determined by Einthoven's method showed a negative deviation in 5 animals (Table III). In man this could be indicative of left ventricular hypertrophy and/or dilatation but whether such a conclusion is justified in the cow remains to be determined. Alfredson and Sykes (1942) and Sellers *et al.* (1958) observed marked variation from animal to animal using the standard bipolar limb leads. From observations on 97 normal dairy cattle, Alfredson and Sykes (1942) noted that the electrical axis of QRS varied within a very wide range although 50 per cent. fell within the values +30° to +90°. It would not be surprising if a left axis deviation did develop at least in some of these cases.

TABLE III
E.C.G. FINDINGS

No. of cases examined	Electrical axis of Q.R.S.				Results indefinite
	+30° to +90°	+120° to +130°	-60° to -80°	-130° to -160°	
9	1	1	2	3	2

Urine Examination

Urine was examined from 3 cases. All samples contained albumen and were negative for sugar and ketones. The *p*H in all the specimens varied from 7.0 to 8.0 despite reduction in appetite. One animal produced up to 12 grammes per litre of albumen in the urine.

J. R. HOLMES—SOME OBSERVATIONS ON TRAUMATIC PERICARDITIS IN CATTLE

FIG. 1.—Case P11, showing marked distension of the pericardial sac with pus and gas and upward and backward displacement of the apical and cardiac lobes of the left lung.

FIG. 2.—Case P1. Pericardium opened showing wire *in situ* and an adjacent penetration but no second foreign body. The wire had impinged on the heart but had not penetrated the myocardium. Note the increased thickness of the pericardium and "cheesy" nature of the pus.

FIG. 3.—Case P9. A typical apex adhesion. The epicardial fibrin has been stripped off.

FIG. 4.—Case P11. The epicardium is coated with smooth fibrin with a constricting effect on the myocardium.

FIG. 5.—Case P3. This shows coarse (sheep's wool type) fibrin and an apex and left side adhesion. The latter has been cut into but the pericardium was firmly adherent to the epicardium with obliteration of the pericardial sac at these sites.

TABLE IV
LEUCOCYTE CHANGES IN BOVINE PERICARDITIS

Duration of illness before first sample	Ref.	Date	Total W.B.C's	Bands	Polys	Lymphs	Monos	Eos.	Bas.	Extent of lesions
Not more than 10 days	P 1	2/2	10,400		9,048	728	312	312	0	
		9/2	10,400	mostly	8,944	936	208	312	0	Uncomplicated
		16/2	11,000	mature	7,370	3,300	220	110	0	pericarditis
		17/2	11,200		7,392	3,360	448	0	0	
Thirty-six hours	P 2	24/6	8,300	2,125	3,995	2,295	0	85	0	Abscesses liver and pleural adh.
Not known	P 3	25/6	11,200	1,904	4,592	4,144	224	336	0	Uncomplicated
Six weeks	P 4	18/10	14,000	mostly	8,260	4,200	420	1,120	0	Uncomplicated
		20/11	11,700	mature	6,669	3,393	468	1,170	0	
At least 14 days ...	P 5	26/6	7,100	213	3,763	2,840	213	71	0	Uncomplicated
Not more than 10 days ...	P 7	22/8	6,200	775	3,007	1,860	527	0	31	Pyaemic absc. kid. pl. adh.
At least three weeks ...	P 8	14/3	8,250	All	5,446	1,402	412	990	0	
		21/3	13,000	mature	8,970	2,470	520	910	130	Uncomplicated
		25/3	16,700		12,191	1,837	1,169	1,503	0	
Very chronic	P 15	10/2	3,500	175	1,505	1,505	35	280	0	Abs. lung. liv. spleen and pl. ad.
	P 13	7/3	5,200	0	1,196	3,276	208	520	0	Idiopath. peri.
	P 16	11/10	10,800	—	4,968	4,320	540	972	0	Tuberculous
		14/10	11,000	—	5,060	4,730	330	880	0	Pericarditis and
		23/10	10,000	—	4,000	5,300	400	300	0	pleurisy
Normal Values Holman (1955)				85·7 ±290	2,029 ±837	3,635 ±1,294	565 ±416	709 ±559	— —	

Haematology

Arthur (1946) reported on the white cell changes in 5 cases of traumatic pericarditis. He found a very marked leucocytosis ranging from 17,000 to 31,000 per cmm., with neutrophilia and a severe shift to the left. Eosinopenia was also a feature particularly in the terminal stages.

The observations on 8 cases of traumatic pericarditis are recorded in Table IV. Fourteen blood samples were collected. Only 1 sample had a total white cell count over 16,000 and the highest total was 16,700 per cmm. In only 3 samples did the leucocytes exceed 12,000 per cmm.

Compared with the values for normal cattle as reported by Holman (1955) in 4 out of the 8 animals the number of neutrophiles in circulation was greater than the mean plus 3 times the standard deviation and in 2 it was increased above the mean plus twice the standard deviation.

In several cases most of the neutrophiles were mature (cases P1, P4, P8). These cows had all shown symptoms for at least 10 days before blood was obtained. Two animals showed a marked shift to the left (cases P2, P3). Of these the first sample from P2 was collected within 2 days of the appearance of symptoms. Unfortunately complete history as to duration of illness was not available for P3.

Two animals died (P4 and P15). In these the eosinophiles were in approximately normal numbers up to the time of death. Both these animals, however, had a history of long-standing serous inflammation. The same applied to case P8 with a history of illness for 6 weeks before death. On the other hand, case P1 reacted as described by Arthur and eosinophiles disappeared from circulation as the disease progressed. Blood samples were examined from 6 cases after rumenotomy. The effect of this operation on the blood picture was not constant.

The case of idiopathic pericarditis (P13) was non-septic and apart from a rather low total count the differential count was normal.

In the animal with tuberculous pericarditis (P16) there was a slight rise in neutrophiles.

Post-mortem **Findings**

Complete *post-mortem* examination was carried out in 14 of the cases in this study. In 11 of these the diaphragm and adjacent pericardium were so adherent that the exact site of penetration of the pericardial sac could not be accurately determined but it seemed likely that in most of them the wire had entered the pericardium in the region of the heart apex. In the remaining 3 penetration had occurred 3 to 4 inches above the heart apex. In 3 cases there was no visible lesion in the mucosa of the reticulum and in one animal slight distortion of the reticular folds was the only indication of the site of puncture. The extent of reticular adhesions to the diaphragm was very variable. In some a thick fibrous cord of scar tissue extended forward from the reticulum to the pericardium. In other cases adhesions were more diffuse, in 2 animals also

involving the adjacent parts of the liver, whilst in 3 animals peritoneal adhesions were very loose in character.

When peripheral oedema was extensive there was usually some excess fluid in the body cavities. In 2 cases there was marked ascites accompanied by oedema of the mesentery.

In all the animals examined the peritonitis was localised and the pericarditis was diffuse. In 2 cases, however, the history and *post-mortem* findings suggested that initial penetration had been followed by localisaton of the pericardial inflammation for some time before it later became diffuse because they showed thickly walled-off pus pockets on the posterior aspect of the heart and more recent inflammation in the other part of the pericardial sac.

The pericardium itself was invariably up to ¾ inch in thickness due to the laying down of fibrin and later fibrosis on its inner aspect (Fig. 2). The character of the pericardial changes is shown in Table V.

In only 3 animals was the heart free, being surrounded by pus and gas. Adhesions of epicardium to pericardium occurred in 11 cases. The sites varied in that the adhesions involved the apex (Fig. 3), the posterior border, right and left sides but the anterior aspect of the heart was always free. They were often so solid that they could not be manually broken down, the myocardium and pericardium being firmly attached to each other by strong scar tissue (Fig. 5). Thus pockets of pus formed around the heart.

The fibrin in the pericardium varied in type. In some cases it was typical "bread and butter" in appearance with strands attaching the layer covering the epicardium to that on the inner spect of the pericardium. In 3 cases it was coarser and more like sheep's wool (Fig. 5) occasionally smooth (Fig. 4) and in long-standing cases fibrous in type.

The character of the pus also varied from being fluid to thick and cheese-like (Fig. 2) and the colour varied from yellow to grey or black. The different types are shown in Table V. In some the layer of fibrin covering the epicardium was only loosely adherent, being underrun by fluid pus. This formed an inner, smaller pus pocket but it was not completely separated from the larger accumulation of pus adjacent to the pericardium. Eleven litres of fluid pus was collected from the pericardium in one case. This gross distension of the pericardial sac resulted in upward and backward displacement of the left lung, particularly the apical and cardiac lobes (Fig. 1). In all except 2 animals, gas escaped when the pericardium was incised.

The foreign body, depending on its length within the pericardium, may impinge on the posterior aspect of the heart or be directed towards the right or left sides. In the majority it either remained posteriorly or towards the left side. In one case the foreign body was on the right side about the junction of middle and upper thirds of the pericardium completely enclosed in the pericardial wall. In only 2 cases had penetration of the myocardium occurred. In one the wire had perforated the left ventricle and in the other an interventricular abscess had opened into the right ventricle with dissemination of infection to the lungs and kidneys.

The lesion in the 8-year-old bull appeared to be very chronic and the wire had penetrated in a more upward direction producing abscesses in the liver and spleen. Thrusting at service may possibly have been the cause. There was no oedema or sign of venous congestion.

Other Lesions

Besides the heart and pericardium and the reticulum-diaphragm the other organ most commonly involved was the lungs and pleural adhesions were most common (Table VI). The pleural adhesions affected mainly the apical and cardiac lobes of the left side and involved adhesions to the underlying pericardium and sometimes also the parietal pleura.

TABLE VI
OTHER LESIONS BESIDES HEART INVOLVEMENT
Traumatic Pericarditis

Number of cases examined	15	
Heart and pericardium only affected	4	
Few areas of pulmonary congestion only	3	
Pleural adhesions, particularly cardiac and apical lobes to pericardium ...	3	
Abscesses liver	1	
Abscesses liver and pleural adhesions	1	
Pyaemic abscesses: kidneys and lungs	1	(R.V. penetration)
Pyaemic abscesses: kidneys and pleural adhesions	1	(L.V. penetration)
Pyaemic abscesses: lungs, liver, spleen and pleural adhesions	1	

TABLE V
POST-MORTEM FINDINGS

Number of cases	Heart free in pericardium	Adhesions of epicardium to pericardium: Apex	Apex and left side	Posterior border	Left and right side	Apex, left and right side	Apex, left and right side and post. border
14	3	1	2	3	1	2	2

Number of cases	Type of pus: Yellow fluid	Yellow granular	Grey fluid	Black fluid	Type of fibrin: Sticky fibrin	Sheep's wool type	Smooth fibrin	Chronic "fibrous"
14	7	1	2	4	3	3	5	3

It is interesting to note that in no case was there any evidence of pulmonary oedema even though very extensive oedema was present in the usual peripheral sites and the body cavities.

All cultures of pericardial pus revealed *Corynebacterium pyogenes* and sometimes also *Escherichia coli*.

Percussion and Auscultation

Percussion

The effect on percussion over the cardiac area depends on the character of the pericardial contents. Where the pericardium was grossly distended by pus and fibrin with resulting upward and backward displacement of the lungs the area of cardiac dullness was increased. If considerable gas formation occurred the area of cardiac dullness sometimes appeared to be decreased in size. Increased resonance on the right side above the point of the elbow was noted if there was a pocket of gas distending the pericardium at that site. Percussion over the cardiac area usually also resulted in grunting due to pain which was more marked if there was an associated pleurisy.

Auscultation

The sites at which auscultation of the heart was carried out on both the right and left sides of the chest were as follows:—

TABLE VII
CHARACTER OF SOUNDS HEARD IMMEDIATELY BEFORE P.M. IN RELATION TO P.M. FINDINGS

Ref. No.	Heart sounds				Post-mortem findings
	L.S. level shoulder	Midway L.S. shoulder-elbow	L.S. int. elbow	Right side midway shoulder-elbow	
P 1	High pitched sounds	Splash	Muffled	Occasional splash	No adhesions. Fluid pus and gas. Cheesy fibrin in lower half of pericardium.
P 2	Snicking sound	Sucking smacking sound	Completely muffled	Fluid splash	Apex and L.S. adhesions. Very little gas. Bread and butter fibrin with "internal" pus pocket.
P 3	Rubbing sound	Muffled but rumbling sound	Completely muffled	Muffled	Apex and L.S. adhesions. 2½ gallons straw coloured fluid. No gas. Sheep's wool fibrin.
P 4	Dullness throughout until rocked, then high pitched (resonant) heart sounds over whole area audible some distance away from animal				Posterior adhesions. 11 litres liquid pus and gas. Bread and butter pericardium.
P 5	High pitched note	Bubbling sound	Mainly muffled but occasional very resonant bubble and splash	—	Posterior adhesions. Grey fluid pus and gas. Little fibrin.
P 6	Heart indistinct, Harsh resp. sounds	Occasional splash with resonance but mainly muffled	Mainly muffled. Occasional sucking sound	Indistinct. Occasional irregular "knock"	Adhesion L.S., R.S. and posterior, with posterior pus pocket. Grey fluid pus. Some gas. Smooth fibrin.
P 7	Heart sounds inaudible, mainly respirations	Distinct but dull	V. muffled. Occasional fluid splash	Occasional tinkling splash, otherwise muffled	Pus/gas pocket on upper right side. Sticky gelatinous fibrin adhesions over remainder of pericardium.
P 8	Fluid and occasional snicking	Rumbling sound	Rumbling sound. Occasional resonance and fluid splash	Muffled but irregular bubbling and fluid splash	Adhesions L.S. and posterior border extending to posterior part of R.S. (post. pus pocket) Yellow pus and gas. Sheep's wool fibrin.
P 9	Heart sounds virtually inaudible except for an occasional sucking sound on L.S. midway between elbow and shoulder				Very little gas. Apax adhesion. Thick yellow fibrin and fluid pus.
P 10	Inaudible L.S. and R.S. Absence of respiratory sounds on R.S. below level of shoulder joint				No gas. Posteriorly towards R.S. thickly walled off pus pocket containing grey pus. Other pus yellow, fluid. Sheep's wool fibrin.
P 11	V. resonant bubbling	Resonant bubbling	Resonant bubbling	Bubble and splash	Gas and fluid pus. No adhesions.
P 12	Fluid splash	V. muffled but occasional tinkling sounds	Fluid splash	Fluid splash	Heart free. Black watery pus and gas.

1. Internal to point of elbow.
2. Midway between point of elbow and level of shoulder, behind the triceps muscles.
3. On level with shoulder joint behind triceps muscles. In the normal animal at this site respiratory (bronchial) sounds predominate.
4. In front of the forearm.

The character of the sounds heard immediately before *post mortem* are considered in relation to the *post-mortem* findings in Table VII.

In 12 animals in which it was possible to correlate heart sounds with the pericardial lesions these had all developed to the exudative stage and the early changes of initial fibrin reaction were not seen on *post-mortem* examination. Three animals were, however, examined clinically in the early stages (P2, P6, P7) and there was a friction rub in 2 and a snicking sound of more sticky fibrin in case P7. Later exudation occurred.

When large quantities of fluid pus accumulated in the pericardial sac without gas, heart sounds became virtually inaudible (case P3 and P10). Very little gas was present in 2 animals on *post-mortem* examination (P2 and P9) and whilst sounds were muffled an occasional splash was audible. When a large amount of gas accumulated under pressure the pericardial sac was distended and drum-like and heart sounds became high pitched and resonant, particularly in the upper part of the area of cardiac auscultation. Furthermore, the auscultation area was extended in an upward and backward direction, heart sounds often being audible above the level of the point of the shoulder, particularly on the left side and in an extended area also on the right side. Where there were no epicardial-pericardial adhesions to form discrete pus pockets, the gas tended to occupy the upper part of the pericardium and heart sounds became resonant in the upper part of the area of cardiac auscultation (case P1, P4, P5, P11, P12). In such cases fluid pus particularly if cheesy in character tended to sink and heart sounds internal to the point of the elbow were usually muffled. At the fluid pus/gas interface splashing was very pronounced (case P1, P5, P12) audible generally at a position approximately midway between the level of the shoulder and the point of the elbow. In the case of P4, complete dullness was a feature until the animal was rocked from side to side when markedly resonant heart sounds became audible over the whole cardiac area. *Post-mortem* examination showed grey fluid pus and gas with little fibrin in the pericardium. Presumably the effect of rocking was to bring the gas bubble into a position between the stethoscope and the heart and thus to increase resonance on auscultation. In the same way fluid splash and bubbling sounds were sometimes audible at one site and then disappeared if the animal altered its position. The abnormal sounds could be restored by moving or rocking the animal (case P4). When splashing sounds were very resonant they could sometimes be heard at some distance away from the animal without the aid of a stethoscope (case P4).

The character of the fibrin may modify the sounds heard. Very coarse fibrin (sheep's wool type) tended to produce a hard rubbing (almost a rumbling) sound as in cases P3 and P8. Early friction sounds were more shuffling in character. The presence of adhesions and pus/gas pockets may modify the sounds very considerably (case P6, P7, P8).

The pathological process is dynamic and changes in the sounds accompanying changes in the character of the contents of the pericardium may occur very rapidly and may vary very markedly at different sites of auscultation.

Other Forms of Pericarditis

Other types of pericarditis encountered in cattle include:—

Tuberculous Pericarditis

One case was seen, the pericardial reaction being mainly dry, characterised by the production of typical granulomata and accompanied by tuberculous pleurisy. There was about one pint of blood-stained fluid in the pericardium. The animal was observed for 12 days. During this time temperature varied from 102.6° to 104° F., pulse from 116 to 140, and respirations from 32 to 64. Heart sounds were virtually inaudible, jugulars were corded, and there was slight oedema in the intermaxillary space.

Idiopathic Pericarditis

One animal was seen with pericarditis the cause of which remained undiscovered. There was no evidence of foreign body penetration. The pericardium was adherent to the epicardium on the left side. On the right side it was thickened and contained odourless red-coloured fluid and large red clumps of coarse fibrin were attached to the epicardium. No growth was produced in culture. Clinically the animal showed corded jugulars and marked oedema in the brisket. On auscultation the heart sounds were rather muffled and appeared irregular in strength but E.C.G. showed no irregularity in rhythm. Pulse rate was over 100.

Adhesive Pericarditis

This has been encountered in 2 animals. This form of pericarditis consists in the development of plastic adhesions between epicardium and pericardium without thickening of the pericardial sac. In these cases it was caused by pressure from without, in one due to exudative pleurisy and the other one had an anterior mediastinal abscess. The pressure on the heart resulted in jugular cording and oedema. In the first case heart sounds were obliterated on both sides of the chest along with respiratory sounds in the lower half on both sides. In the cow with the mediastinal abscess the heart was displaced upwards. Heart sounds were inaudible in the lower third of the chest but could be heard on a level with the shoulder. Pulse rate in both animals was over 100.

Discussion

In traumatic pericarditis the typical foreign body is the piece of wire or nail often 2½ to 4½ inches long penetrating from the reticulum through the diaphragm. Results of reticular penetration are extremely variable, not only in their sequelae but also in the time relation between initial penetration

and subsequent effects. It is difficult to estimate how many wires which perforate the reticulum would enter the pericardium. The actual proportion is probably not high, but all are potential causes of pericarditis and of all the consequences of foreign body ingestion penetration of the pericardium is one of the most serious.

When an increase in heart rate occurs the possibility of primary circulatory dysfunction should always be considered. The rise in the pulse rate was found to be valuable clinical information in the diagnosis of pericarditis. This agrees with the observations of other workers (Arthur, 1947; Blood & Hutchings, 1955b). In contrast body temperature was very variable and proved of no value as a diagnostic aid.

Reticular penetration is usually rapidly followed by fibrinous adhesion between reticulum and adjacent diaphragm to wall off spread of infection and the resulting peritonitis is generally localised. It is reasonable to suppose that the same type of defence reaction occurs when a foreign body enters the pericardium. The constant movement of the heart, however, and additional reticular and diaphragmatic movements in many cases probably serve to force the foreign body further into the pericardial sac and the resulting pericarditis tends to be diffuse.

Auscultation provides the best method of establishing a diagnosis and further confirmation may be obtained if necessary by pericardial puncture. The heart sounds are modified by a variety of factors. The pathological process is constantly changing. Consequently sounds vary as the process progresses and also according to the site of auscultation. Of particular importance is:—

(1) The presence of gas which determines whether splashing may occur. Splashing sounds do not occur in the absence of gas and if fluid accumulates when gas is not present heart sounds tend to become muffled.

(2) The type of fibrin. Coarse fibrin produces a rumbling sound, fine fibrin a shuffling noise. These are synchronous with the heart sounds. Where localised pleurisy overlies the pericardium the sounds produced by movement of the adherent lung may be synchronous with the heart beat. Generalised fibrinous pleurisy produces sounds synchronous with the respirations. Friction sounds may have to be differentiated from the murmurs of incompetent or stenotic valves (endocarditis), but are usually characterised by a double note whilst cardiac murmurs are generally single sounds.

(3) The character of the pus. Cheesy pus tends to sink resulting in dullness in the lower part of the area of auscultation. Gas tends to rise producing increased resonance in the upper part of the area which often extends on the left side to the level of the shoulder joint or above.

(4) The presence of pericardial adhesions and the formation of pockets of pus and gas may modify the sounds very markedly.

The initial pericardial penetration is generally accompanied by a fibrinous reaction and the production of typical friction sounds. This is usually followed by exudation. The speed with which exudation occurs is very variable. Evidence from this study indicated that it may occur very rapidly. Thus when first seen the early friction sound may have been replaced either by obvious splashing or muffled heart sounds.

Haematology provides information on the presence of a pyogenic focus and in conjunction with the clinical findings is supporting evidence for a diagnosis of traumatic pericarditis. The high total counts reported by Arthur (1946) were not observed in this study. Like the pathology the blood picture is dynamic being influenced by the stage and age of the pericardial lesion and possibly also by the extent of involvement of other organs.

Thirteen of the animals considered in this paper were destroyed. A notable observation was the fact that most were quite bright and still ruminating and eating despite the fact that the heart was surrounded by pus and there was gross peripheral oedema. Whilst it is probable that some affected animals die of toxaemia the suggestion is advanced that many would die from mechanical interference with heart action due to excessive fibrosis in the pericardium. The *post-mortem* findings in 2 cases particularly were indicative of a long-standing localised infection which was later aggravated by extension to involve the whole pericardium. The thickened pericardium probably acts as a barrier not only to the spread of infection in the chest but also to the absorption of toxins. The character of the pus and gas surrounding the hearts of most of these animals was such that one would have expected them to have been severely toxic.

The reticular penetration in the bull was atypical in that the foreign body was directed upwards possibly during service. Consequently there was no reaction to the usual clinical tests for foreign body penetration.

An interesting feature of heart disease in the cow is the frequent absence of oedema in the limbs, transudate accumulating particularly in the loose connective tissue of the neck and ventral aspect of the sternum. In all these cases there was complete absence of pulmonary oedema even when there was very severe transudation elsewhere. It is reasonable fo assume that the vascular congestion is caused by the constricting effect of the pericardial exudate on the heart. The inflammatory reaction is usually diffuse. The effect will be greater on the thin-walled right ventricle than on the thicker walled left ventricle. Thus blood will tend to be dammed-up in the systemic circulation. Furthermore, left ventricular hypertrophy would tend to overcome increasing systemic peripheral resistance and blood is therefore less likely to be held back in the pulmonary circulation at least initially.

Even with severe pericarditis 4 cases failed to develop peripheral oedema. No association was found between the site of epicardial/pericardial adhesions and the development of oedema and it is considered that the factor which influenced the degree of circulatory congestion (and thus peripheral transudation) was the rate of fibrosis in the pericardium. If the lesion develops slowly the heart

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An Aspect of Infertility in the Dog

BY

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Introduction

OVER the period of the last 5 years this Department has been asked by veterinary surgeons in practice to carry out the collection and examination of semen from many dogs which were not achieving satisfactory results at stud.

In some instances a microscopical examination of the semen revealed that it was normal, and the poor conception rate was attributed to either mismanagement at stud or infertile bitches. Latterly, however, a condition has arisen which is causing grave concern to many breeders and veterinarians.

History

The picture is similar in almost all cases—the variations being the age and breed of the dog. The dogs examined have all been pedigree types and many of them have achieved high distinction in the show ring. All have been proven stud dogs which for some unaccountable reason have become sterile. In some cases this sterility has been a gradual process, but in the majority it has occurred suddenly with little or no warning.

Most owners on being questioned told a similar story—that the dog had been introduced gradually to stud work at about 10 to 12 months of age, and had only served very few bitches during its first season. On gaining recognition in the show ring they had naturally been in greater demand and had subsequently been mated more frequently.

All had been extremely successful at stud until suddenly bitches had failed to conceive to them. On inquiring into the history of the bitches served it was interesting to learn that most of them subsequently conceived at their next heat when served by some other stud dog, and thus one could remove the suspicion that the bitches might be at fault.

It was also interesting to note that all the dogs still continued to serve willingly and without any inhibitions in spite of their apparent sterility.

Symptoms

A clinical examination of the dog was carried out in every case before any semen collection was made. Some of the dogs were suffering from atrophy of one or both testicles. This condition appeared to cause no pain and did not interfere with the libido of the dogs.

Bacterial examination of preputial swabs revealed nothing abnormal.

The collection of a semen sample was carried out using a canine artificial vagina in all cases, the semen obtained being immediately examined microscopically (see Table I).

Treatment

This was usually left to the veterinary surgeon in charge of the case, but in 4 cases advice was given and treatment carried out. This consisted of bi-weekly intramuscular injections of both follicle stimulating hormone and luteinising hormone in doses of 100 I.U. The administration of these gonadotrophic hormones was carried out in the hope of improving both the quantity and quality of the ejaculates in a similar manner to that used to improve the fertility of rams (Parkes & Hammond, 1940).

No improvement was observed in any of the cases treated, although treatment was continued for a period of 1 month and the dogs examined 2 to 4 weeks after it ceased (see Table I).

Unfortunately it was not possible to carry out *post-mortem* examinations on any of the animals, for, because of their value, owners insisted on keeping the dogs in the hope that time and rest might in some manner aid recovery. Up to date no reports have been received that have justified such expectations.

Discussion

On reviewing relevant literature no mention is

Some Observations on Traumatic Pericarditis in Cattle. —*Concluded.*

may be better able to accommodate than when fibrosis rapidly constricts the myocardium.

Jennings and McIntyre (1957) reported a successful pericardectomy operation for the relief of pericarditis in a heifer. It is worth noting that in this study only 3 hearts out of 14 were free in the pericardial sac on *post-mortem* examination. Extensive pericardial adhesions would seem to present a very serious obstacle to the successful practice of this surgical technique.

Acknowledgments.—I wish to express my sincere thanks to Professor C. S. G. Grunsell for help in the compilation of this paper. The invaluable help of colleagues in the departments of Veterinary Medicine and Veterinary Surgery in the University of Bristol is gratefully acknowledged. I wish to thank Mr. R. S. Undrill for the photographs and the numerous veterinary surgeons in practice who kindly provided the clinical material for this study.

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made of any similar condition to the one described here. Bloom (1954) discusses atrophy of the testicles in the dog but states it is usually encountered in dogs over 10 years old and is due to senility. Other causes of atrophic testicles are orchitis or tumour formation, neither of which conditions were encountered in the dogs examined here nor could any history relating to such conditions be traced.

One is left wondering quite how widespread this condition may be, for obviously owners of valuable stud dogs are reluctant to admit that one of their

(*Concluded at foot of page* 364)

TABLE I

Breed of dog.	Age of dog	Whether exhibited at shows	Findings at clinical examination	First Microscopical Examination of Semen				Colour of ejaculate	Treatment (if any)	Whether examined subsequently at 6–8 weeks	Second Microscopical Examination of Semen			
				Volume	Density	Motility	Morphology				Volume	Density	Motility	Morphology
	yrs.			ml.							ml.			
Corgi	4	Yes	Normal	2·0	No sperms present			Clear	None	No				
Corgi	3	,,	Right testicle slightly atrophied	2·7	,,		,,	,,	Bi-weekly injection of F.S.H. and L.H. 100 I.U. of each	Yes	3·1	No sperms presemt		
Beddlington terrier	5	,,	Normal	4·2	,,		,,	,,	As above plus ½ gr. Thysoil tablets daily	,,	5·6	,,		,,
Norwich terrier	4	,,	,,	2·5	,,		,,	,,	None	No				
Dachshund	3½	,,	,,	1·7	,,		,,	,,	,,	,,				
Dachshund	4	,,	Both testicles very small	2·8	,,		,,	,,	,,	Yes	2·5	,,		,,
Poodle (min.)	2½	,,	Right testicle slightly atrophied	3·2	,,		,,	,,	,,	No				
Yellow Labrador	7	No	Normal	9·4	,,		,,	,,	,,	,,				
Dachshund (min.)	2½	Yes	,,	3·1	,,		,,	,,	Bi-weekly injection of F.S.H. and L.H. 100 I.U. of each	Yes	3·2	,,		,,
Pekingese	3	,,	,,	3·7	,,		,,	,,	,,	No				
Norwich terrier	3	,,	,,	4·1	,,		,,		,,	Yes	4·9	,,		,,
Dalmatian	4½	,,	,,	6·2	,,		,,	,,	None	No				
Greyhound (ex-racing)	4	No	,,	13·2	,,		,,	,,	,,	Yes	11·9	,,		,,
Bloodhound	5	Yes	,,	9·7	,,		,,	,,	,,	,,	10·7	,,		,,
Bassett hound	4½	,,	,,	8·6	,,		,,	,,	,,	,,	7·7	,,		,,
Poodle (toy)	3¼	,,	Both testicles slightly atrophied	3·1	,,		,,	,,	,,	Yes	2·1	,,		,,
Alsatian	4	No	Normal	5·4	,,		,,	,,	,,	No				
Poodle (min.)	3¼	Yes	,,	3·1	,,		,,	,,	,,	,,				
Poodle (toy)	2½	,,	Left testicle slightly atrophied	2·7	,,		,,	,,	,,	,,				
Bulldog	4½	,,	Normal	4·6	,,		,,	,,	,,	,,				
Pekingese	3½	,,	,,	3·2	,,		,,	,,	,,	Yes	1·7	,,		,,
Poodle (toy)	3½	,,	Normal	3·7	,,		,,	,,	,,	,,				
Schnauzer	2¾	,,	Both testicles slightly atrophied	4·4	,,		,,	,,	,,	,,	4·6	,,		,,
Dachshund (min.)	3	,,	Normal	2·9	,,		,,	,,	,,	No				
Poodle (min.)	3½	,,	Right testicle slightly atrophied	2·3	,,		,,	,,	,,	,,				

Some Observations on the Effects of Changes in Environmental Conditions and Diet in Ewes

BY

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SUMMARY.—Turning ewes from loose-boxes to a permanent pasture was found to result in a decrease in their plasma magnesium and calcium concentrations, while their plasma inorganic phosphate concentrations increased. A move from permanent pasture to a clover-mustard ley also resulted in a decrease in plasma magnesium concentrations. In this case inorganic phosphates decreased, while the calcium concentrations increased. No clinical abnormalities were detected. These findings are compared with those resulting from similar changes of pasture previously reported in the literature.

Introduction

THERE have been several reports in the literature of "grass tetany," or hypomagnesaemia, in sheep and it seems to be generally agreed that its incidence is increasing (Stewart, 1954; Penny & Arnold, 1955; Hughes & Kershaw, 1958). Most reports described the clinical condition and include the results of the biochemical examination of samples of blood taken from affected sheep. Alternatively blood has been taken from clinically normal sheep from the same flock, on the assumption (Stewart, 1952) that clinical cases occur only when there is a general hypomagnesaemia in the flock. The disease is most commonly seen after a move from an old pasture, or rough grazing, to a young rapidly growing grass ley in the spring. It is most commonly seen, therefore, in heavily pregnant ewes or in those with lambs at foot. Clinically the condition is essentially a hyperexcitability, which may increase in intensity and be followed by tetany and death.

Blumer, Madden, and Walker (1939) described a similar condition in Australia in breeding ewes on lush pasture, which was usually precipitated by droving following the winter rains. Biochemical estimations on blood samples showed hypocalcaemia, together with hypermagnesaemia, although no figures are quoted. (They refer to "hypocalcaemia or grass staggers," but it seems preferable to keep the term grass staggers for the condition where a hypomagnesaemia can be demonstrated, as with cattle.)

Stewart (1952, 1954) reported hypomagnesaemia among both hill and lowland ewes in Scotland in the spring. Pook (1955) described an outbreak in the South of England in a flock of recently lambed ewes, following a move to a first-year ley. Serum samples showed hypomagnesaemia, but normal serum calcium concentrations. Penny and Arnold (1955) described an outbreak occurring under very similar conditions, also after a change of pasture. Affected ewes showed very low serum calcium, magnesium and inorganic phosphate concentrations. Hughes and Kershaw (1958) described 2 outbreaks among ewes in Wales which had recently been brought off the hill to lowland pasture for lambing. Clinically the two conditions appeared to be essentially the same, although on biochemical examination of serum one proved to be a simple hypomagnesaemia while the other was a mixed hypomagnesaemia and hypocalcaemia.

Inglis, Weipers, and Pearce (1959) have recently published the results of their work on the influence of environmental factors and short starvation on the serum magnesium concentration of sheep.

In all the outbreaks described above the biochemical estimation of blood constituents from affected ewes, or from ewes in a flock where the condition had occurred, appears to have been confined to one sample per animal. Although the onset of the clinical disease is rapid, it is not known whether the change in the serum concentrations is similarly rapid, or whether it is a more gradual process. In addition there is little information available on how quickly the subclinical condition of hypomagnesaemia resolves spontaneously.

For this reason, some of the ewes on the Veterinary School farm were in one case moved from loose-boxes

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An Aspect of Infertility in the Dog.—*Concluded.*

best stud dogs has become sterile for fear that the dog-breeding public will instantly infer that some infectious disease is affecting the whole kennel and that therefore they will lose a considerable amount of business.

The information available gives no definite indication as to the aetiology of the condition. The histories of the affected animals suggests that inherited factors may be involved because in several cases ancestors were said to have been similarly affected and in the immediate post-war period there was a considerable amount of in-breeding and in some cases selection for characteristics such as small size which may have involved involuntary selection for testicular abnormalities. There is no evidence to suggest that nutritional deficiency could be responsible, but other environmental factors such as large numbers of services over a prolonged period may well have played a part. It may be also that the testicular damage is the aftermath of an unrecognised orchitis or epididymitis. The elucidation of the causes of the condition depends on further, more detailed investigations.

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to pasture, and in another case from a permanent pasture to a young grass ley (in spring in each case), and any changes in their plasma calcium, magnesium and inorganic phosphate concentrations which resulted were noted.

Materials and Methods

Clinically normal Dorset Horn ewes were used and blood samples taken from the jugular vein into 30-ml. glass bottles, using solid Heparin as anticoagulant. The blood was centrifuged and plasma obtained within 1 to 2 hours.

For the estimation of plasma calcium and magnesium concentrations a modification of the method of Wilson (1955) was used. Two-ml. samples of plasma were taken and calcium was precipitated as calcium oxalate which was then centrifuged. The supernatant was decanted and magnesium precipitated as magnesium ammonium phosphate, which was also centrifuged. Both precipitates were dissolved in 2 drops of 50 per cent. v/v nitric acid and titrated with Ethylene Diamine Tetracetic Acid, after buffering with Ethanolamine Hydrochloride (*p*H 10.4), using Solochrome Black as the indicator.

Plasma inorganic phosphate was estimated using the ammonium vanomolybdate method of Simonsen, Wertman, Westover, and Mehl (1946).

Experiment 1. Four ewes had been maintained in 2 loose-boxes for about a month (February 7th to March 3rd, 1958), and for the latter part of this period they had been bled daily. Two of these ewes (Nos. 9 and 43) were pregnant. They were given kale, crushed oats, and hay with water *ad lib.* On March 3rd they were turned to a small permanent pasture (rye grass and timothy mixture). The changes in plasma calcium, magnesium and inorganic phosphate concentrations which resulted are shown in Fig. 1.

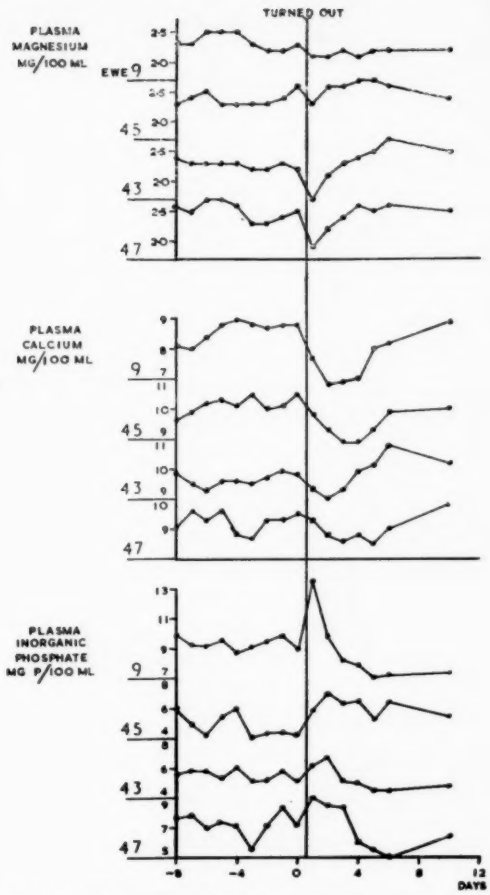

FIG. 1.—The effect of turning ewes from loose-boxes to pasture in spring.

It will be seen that the move from loose-boxes to pasture resulted in a fall in the plasma magnesium concentration in all 4 ewes within 24 hours. This was most marked with ewes 43 and 47, with a decrease from 2.2 to 1.7 and from 2.5 to 1.9 mg. per 100 ml. respectively. During the next 24 hours all values tended to increase again towards normal, excepting that of ewe 9 which had shown least decline, and which remained steady during this period.

A fall in the plasma calcium concentration accompanied that of magnesium, most marked in ewes 9 and 45, which had shown least change in magnesium concentration. Inorganic phosphate concentrations increased, particularly in ewes 9 and 45. Ewe 9 showed an increase from 9.0 to 13.6 mg. P per 100 ml. during the first 24 hours. After 48 hours it had fallen again to 9.9 mg. P per 100 ml.

It will be seen, then, that the ewes which showed the most marked fall in plasma magnesium concentration, ewes 43 and 47, were those in which there was least change in plasma calcium and inorganic phosphate concentrations. The other two ewes, 9 and 45, showed only slight falls in plasma magnesium, but more marked change in plasma calcium and inorganic phosphate concentrations. The inverse relationship between plasma calcium and inorganic phosphate is well illustrated. It is also interesting to note in ewe 9 that the plasma phosphate concentration, after its initial rise, had returned within normal limits by the second day at pasture. The plasma calcium concentration, on the other hand, remained at a low level until the fifth day.

Experiment 2. A group of ewes were moved from the permanent pasture where they had been since lambing, about a month previously, to a young clover-mustard ley on April 21st, 1958. The effects of this move on the plasma concentrations of calcium, magnesium and inorganic phosphate were followed in four of the ewes, all of which had been bled 11 days previously and 3 of them 10 days prior to this. In the experimental period, the first blood sample was obtained about one hour after the change of pasture (warning of the move had not been given). One of the ewes (43) was involved in both of the experiments. The results are shown in Fig. 2.

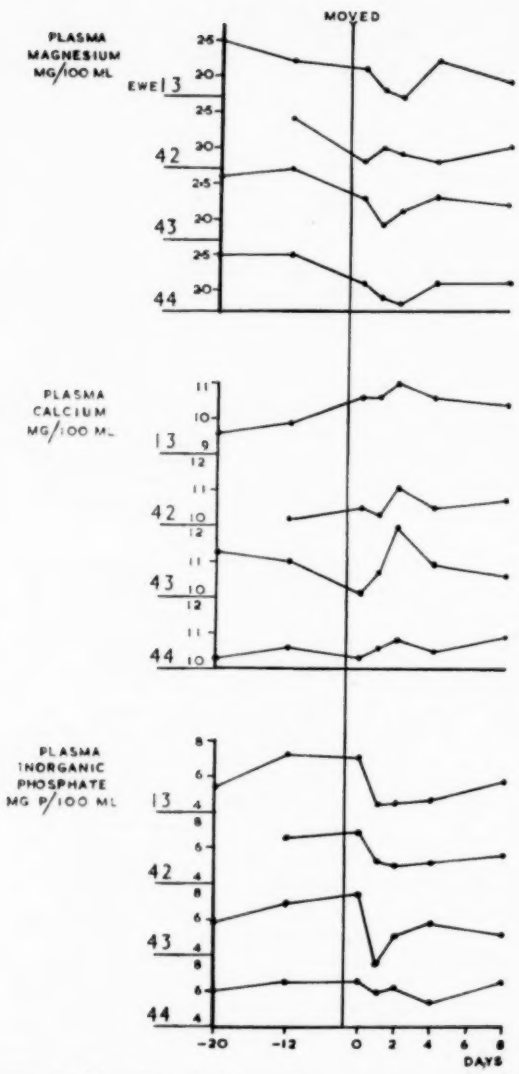

FIG. 2.—The effect of change of pasture on ewes in spring.

When sampled one hour after the change of pasture, plasma magnesium of all the ewes showed a decrease in concentration since the previous bleeding. Ewe 42, which showed the most marked decrease during this period, from 2.3 to 1.8 mg. per 100 ml., was the only one not to show a further decrease during the 24 hours after the move. Ewes 13 and 44 showed a falling magnesium concentration up to 48 hours, with values as low as 1.7 and 1.8 mg. per 100 ml. respectively.

Unlike Experiment 1, when the reverse changes occurred, there was a tendency for the plasma calcium concentrations to increase, while the inorganic phosphates decreased, following the change of pasture. This was most marked in ewe 43 with plasma calcium increasing from 10.1 to 12.0 mg. per 100 ml. within 48 hours, while the plasma inorganic phosphate fell from 7.4 to 3.5 mg. P per 100 ml., a decrease of over 50 per cent., within 24 hours. Both ewes 13 and 42 showed similar, although less marked, changes in inorganic phosphate concentration, which gradually increased again during the next eight days.

Discussion

The present work has shown that moving the ewes in one case tended to result in a decrease in plasma concentration of both magnesium and calcium, while the inorganic phosphate tended to increase. In the other instance the plasma magnesium again decreased, but this time the plasma calcium tended to increase, while 3 of the 4 ewes showed a marked fall in inorganic phosphate concentration. In each experiment the general trends for all the ewes in that experiment were the same, although there was a variation in degree among individual ewes. As a generalisation, those ewes which showed most marked change in plasma magnesium showed least change in calcium and inorganic phosphate concentrations, and *vice versa*. In no case were clinically detectable abnormalities noted, although it is reasonable to assume that exacerbation of these trends could result in the precipitation of hypomagnesaemic tetany.

Ewe 43, which was involved in both experiments, is interesting in that on each occasion a fall in plasma magnesium concentration occurred. In Experiment 1, however, calcium and inorganic phosphate were little affected, while in Experiment 2, the change in these was marked.

The variation of "normal" values for individual sheep is also illustrated. Ewe 9 (Experiment 1) had a plasma calcium which varied between 8 and 9 mg. per 100 ml. when indoors, while that of ewe 45 was normally between 10 and 11 mg. per 100 ml. This variation (but in a reciprocal manner) was even more marked with inorganic phosphate concentrations, with corresponding values of 9 to 10 and 4 to 6 mg. P per 100 ml., respectively. A value of 4 mg. P per 100 ml. is therefore quite normal for ewe 45 but would be low for ewe 9.

Franklin (1948) showed that moderate exercise could result in a fall in the plasma calcium concentration of normal sheep, and could also give rise to symptoms in ewes which had been hypocalcaemic, but clinically normal, prior to the exercise. It seems likely that the increase in exercise in the present

PLASMA CONCENTRATION

Mg.	*Ca.*	*In.P.*	*Author*
+	−	N.R.	Blumer, *et al.* (1939).
−	0	N.R.	Pook (1955).
−	−	−	Penny and Arnold (1955).
0	−	N.R.	Hughes and Kershaw (1958).
−	−	N.R.	Hughes and Kershaw (1958).
−	−	+	Present Experiment 1.
−	+	−	Present Experiment 2.

Key: 0 = No change.
+ = Increase in concentration.
− = Decrease in concentration.
N.R. = Not recorded.

(*Concluded at foot of col.* 1, *page* 367)

The Prevention and Treatment of Rupture of the Perineum in the Cow*

BY

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RUPTURE of the perineum involves a traumatic upward extension of the vulval orifice towards, and sometimes into, the anus. It is one of the hazards of parturition and is important because it often leads to infertility.

Causation

The condition arises when natural (parturient), or artificial (obstetrical), force causes the presenting foetus to overdistend the vulva; the engaging foetal parts may be too large, the vulva too small, or the force may be at fault. It is thus due to developmental errors or obstetrical mismanagement. Largeness of the foetus and smallness of the vulva occur as developmental abnormalities which are probably governed by genetic and endocrine factors; for instance, oversized foetuses and hypoplasia of the vulva are seen particularly in Friesian cattle, and this is the breed with the highest incidence of perineal rupture. (Constriction of the vulva can, of course, follow trauma at a previous parturition.) Obstetrical mismanagement can affect both foetus and vulva; for example, in cases of obstetrical delay the foetus may undergo emphysematous enlargement and the vulva may contract due to the recession of parturient hyperplasia. It may be noted, in passing, that obstetrical delay also predisposes to perineal rupture by reducing lubrication and by allowing uterine inertia to develop; consequently, inordinate and artificial force is required for delivery.

Another obstetrical error which contributes to perineal rupture is too hasty traction on a normally presented foetus before the vulva has undergone gradual dilatation by the foetal "wedge." It is evident, also, that the application of traction to a wrongly disposed foetus could cause the condition. Such obstetrical errors should not occur under veterinary care. Although perineal rupture may arise spontaneously, it usually occurs when traction is being applied.

Prevention

In considering the foregoing causes of rupture of the perineum, it will be seen that prevention of the condition involves genetic as well as obstetrical considerations but the former are difficult to apply. It becomes mainly, then, a matter of correct obstetrical practice. Thus grossly oversized calves should be delivered by caesarean section, or following embryotomy. In the less severe cases, where traction is indicated, the application of force must be moderate and synchronous with the maternal efforts. Gentleness and patience are prime requisites of the obstetrician. In delayed cases lubrication is essential. In no circumstances must an attempt be made to deliver by traction an improperly disposed calf before correction of its presentation, position, and posture.

Notwithstanding the foregoing advice, veterinary surgeons will occasionally meet cases in which, despite the accepted precautions, it will be found impossible to proceed with parturition without perineal rupture occurring. This position arises when the foetal occiput engages the vulva in cases of relative foetal oversize and in heifers with vulval hypoplasia. In these animals, if after proper lubrication and continued patient effort, the vulva will not extend further, episiotomy should be performed.

Episiotomy. This entails incising, in an archiform manner and in a dorso-lateral direction, each vulval labium in its upper third. Cutting directly upwards into the perineal raphe is wrong because, once started, further birth of the foetus will tend to cause a traumatic extension upwards and sometimes into the anus. (Friermuth, 1948). The requisite depth of the vulval incisions can be decided only by trial on the basis of the minimum amount to allow

* Presented to a meeting of the Central Veterinary Society on November 12th, 1959.

Effects of Changes in Conditions and Diet in Ewes.—*Concluded.*

Experiment 1, after the move from loose-boxes to pasture, may have contributed to the hypocalcaemia which resulted, while the hypomagnesaemia is more likely to have been dietary in origin. Exercise was almost certainly the important precipitating factor of the hypocalcaemia in the cases reported by Blumer *et al.* (1939), and was probably of importance also in the cases reported by Hughes and Kershaw (1958).

Why a change in environmental or dietary conditions should have such varying results in different instances is not clear, although it suggests that a number of factors are involved. The changes which have been recorded in the literature already cited and also in the present experiments are listed below.

Acknowledgments.—This work was undertaken during the tenure of a Research Training Scholarship awarded by the Animal Health Trust. The author would also like to thank Professor W. I. B. Beveridge for providing research facilities, Mr. A. A. Wilson for advice and encouragement, Mr. R. Kimberley for technical assistance, and Miss J. Minns for the photographs.

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delivery. By gentle traction on the foetal head so as to cause firm engagement of the occiput in the vulval orifice, it is easy to ascertain the necessary depth of incision. Local infiltration anaesthesia, or epidural block, are required and, immediately following delivery, the wounds are sutured with vertical mattress sutures of 15-lb. braided nylon. The wounds heal readily.

Repair of Ruptured Perineum

The best time to treat perineal tears is as soon as possible after parturition—with a view to promoting first intention healing. Posterior epidural anaesthesia is induced, the parts washed, and debridement carried out. The wound is then sutured with deep vertical mattress sutures, beginning at the bottom of the wound, and tying the sutures only when all are placed. A large full-curved needle, and needle holders are essential. It is surprising how well these wounds heal.

If more than 2 days have elapsed since parturition, cicatrisation must be allowed to occur. Owing to a failure of vulval apposition, atmospheric air now passes in and causes ballooning of the vagina; chronic vaginitis, usually with infertility, results. There is now the choice of intra-uterine insemination, thus by-passing the vaginal infection, or of reconstructing the perineum, using the technique described by Gotze (1938). I cannot improve on Gotze's technique nor on his description of the method. He prepared an excellent film of his operation before the war. But I can emphasise some of the technical points:—

1. A cow should not be operated on when in oestrus.

2. The operation should be performed on the standing animal under the influence of chloral hydrate sedation and posterior epidural anaesthesia.

3. It is essential to cut forwards as far as possible in the horizontal plane *i.e.* to dissect away all scar tissue from the rectal floor.

4. Before, and during, the placing of the sutures the rectal floor should be firmly retracted.

5. The sutures should be of No. 4 catgut. All should be placed before any are tied. A large curved needle is essential. It is important to understand what is required of the sutures and to realise that they are mattress sutures designed to bring together the denuded tissues. They must be tied tightly.

The skin sutures are of braided nylon and should be removed in a week.

A normal diet is indicated; constipation, or diarrhoea delays healing.

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Extirpation of the Anal Sacs of the Dog*

BY

M. YOUNG

Ealing

IT may seem strange that this straightforward operation for the extirpation of the anal sacs in the dog should be the subject of a paper, but I believe that this operation is not very often performed in veterinary practice. In my view, this is so because of a considerable prejudice against it. The text-books inform us of the extensive plexus of blood vessels supplying the anal regions, conjuring up pictures of severe and uncontrollable haemorrhage. Many of us have been told of cases where the operation has resulted in damage to the nerves supplying the anus, and I, myself, have heard a client describe in harrowing detail how her dog had had little control on defaecation after undergoing this operation; and also tales of wounds which would not heal, causing pain and suffering to the unfortunate pets. It was thus several years after qualifying before I attempted the operation myself. I was agreeably surprised to find first intention healing after 7 days; the patient did not worry about his wounds, the results were excellent, the client was grateful, and there were no complications. Perhaps it was as well that I did not perform this operation during my early days in practice, for I believe the task is one for the experienced and competent surgeon who is used to using a knife in a confined space and only cutting that which he intends to cut.

Although the operation is simple and straightforward it is essential that the dog should be properly positioned, and the operation must be carefully planned. It may well be that many surgeons have carried out the operation initially believing it to be a very minor and simple operation, and this may account for some of the disastrous results that have been reported. It is, of course, common practice to empty the anal sacs by means of digital pressure, and to treat inflammation of their lining by the insertion of antibiotics or caustics. Whilst I frequently resort to all these measures, it is my view that when the sacs are a constant source of irritation and pain to the patient the obvious and best treatment is their surgical removal. I fail to see any reason against this procedure as I consider that the sacs are of no value to the domestic pet.

The Operation

Prior to the operation the patient is starved, premedicated, and anaesthetised to the plane of deep surgical anaesthesia. It is placed in the prone position, with the hind quarters raised and the tail well pulled forward by a tape. The perineal region is then clipped, shaved, and sterilised. Whilst the anal sacs lie ventro-lateral to the anus, the ducts enter the anal ring in the lateral position. Each sac is now filled through its own duct so that the gland can be clearly felt, and in small dogs it can

* An address to the Central Veterinary Society, November 12th, 1959.

be seen through the skin. I usually use damp cotton wool, which I insert easily with the aid of a probe; others prefer to use wax or plaster of Paris, while some use nothing at all. A dorso-ventral incision is then made over the sac and usually about ½ inch from the anal rim; this is deepened through the subcutaneous tissue and, with care, through the muscle covering the external aspect of the sac. The sac can be readily distinguished from its surrounding tissue by its dark grey colour, and once it is reached it is very carefully dissected free of the surrounding muscle, especial care being taken not to incise the sac. When the external aspect has been adequately exposed the sac is grasped with allis forceps and then pulled through the skin incision whilst the tissues on its under aspect are dissected away.

During this phase of the operation a scalpel carrying a No. 15 Paragon blade is used, holding the blade parallel to the external surface of the sac; in this way there is less danger of cutting any of the surrounding blood vessels. But should this occur haemorrhage can easily be controlled with the use of artery forceps and later ligation. The sac is now attached to the anal ring by its duct and, at this stage, it is important to release the tension applied with the allis forceps, and to cut through the duct as it leaves the anus, not making the mistake of removing with it part of the rectal wall. As a guard against the latter a finger is inserted into the anus while the duct is severed. Should a portion of the rectal wall be accidently removed, it can be ligatured with fine mattress stitches of 000 catgut, but if the duct has been severed correctly I do not think it is necessary to ligate the duct or stitch the rectal wall in any way. It is now most important to close the dead space left after the removal of the sac, and this is done with 3 or 4 deep interrupted catgut sutures bringing together the muscle tissue on either side of the wound. Lastly, the skin incision is closed with 3 to 6 interrupted sutures of strong Vetafil. The same procedure is used to remove the second sac.

Post-operative Treatment

In my experience very little after treatment is required, although a highly strung dog may need to be tranquillised. First intention healing occurs almost without exception, and the sutures can be removed 7 to 10 days after the operation.

THE REGISTER OF VETERINARY SURGEONS

Mr. Walter John Ehret, B.V.SC. (PRETORIA), c/o South Africa House, Trafalgar Square, London, S.W.1, was registered in the Commonwealth List of the Register on April 28th, 1960.

Erratum

It is regretted that in last week's issue the name of Professor G. Payling-Wright was mis-spelt Tayling-Wright. We tender our apology.

Current Literature

BOOK REVIEW

Advances in Veterinary Science. Vol. V. Editors: C. A. BRANDLY and E. L. JUNGHERR. Academic Press Inc., New York and London (1959). $14 (approximately £5).

It is to be noted from the fore-matter of this volume that the vacancy on the Advisory Board caused by the death of Professor A. Hjärre has not been filled.

The first volume of the series was published in 1953 and the efforts of the Editors in producing a total of 5 volumes in a period of 6 years are to be commended.

In this, the fifth volume, there are 7 contributors, compared with 21, 11, 10, and 11 in volumes I, II, III, and IV respectively. The present volume has 450 pages, the preceding volumes varying in size from 414 to 579 pages.

As will be seen from the detailed consideration of the individual chapters, the range of subject matter in the present volume is wide and the choice of contributors is world wide.

The first chapter, entitled "Examination of the Eye and Eye operations in Animals," is from the pen of O. Uberreiter of the Clinic for Surgery and Obstetrics, Veterinary College, Vienna, Austria. The chapter extends to just over 72 pages with rather more than 7 pages of references covering publications from many countries. Examination of the eye is dealt with in 32 pages. Many of the techniques described can be carried out by general practitioners with relatively simple equipment, and there is nothing very novel in any of those described. Some more elaborate techniques are described in detail; these require special apparatus unlikely to be found other than in a very fully equipped veterinary hospital.

The second part of the chapter dealing with eye operations is detailed but not unnecessarily so. There are a number of excellent illustrations in this part of the chapter. The limitations on eye surgery in animals are indicated by a sentence in the brief section on retinal detachment which states "operations (for retinal detachment) are not practical in veterinary patients."

The next chapter on "Q Fever: A Zoonosis," is contributed by B. Babudieri of Instituto Superiore di Sanità, Laboratorio di Microbiologia, Rome. The text of the chapter extends to just over 73 pages, but there are rather more than 28 pages of references. An "Historical Outline" is followed by a lengthy consideration of the geographic distribution, which certainly establishes that the disease is extremely widespread. The aetiologic agent, *Coxiella burnetii*, is considered in some detail as are the various vectors. The disease in wild animals, in domestic animals, and in man, receives attention, after which the author reverts to the mechanism of contagion. Methods of diagnosis are discussed in considerable detail. Therapy receives but brief consideration because "there have been very few reports of satisfactory treatments of Q fever with drugs other than the antibiotics." The antibiotics that have proved most

useful are chloramphenicol and the tetracyclines. Finally, control measures are outlined and the shortcomings of vaccines emphasised. The section headed "Conclusions" contains some interesting statements. "Q fever is certainly, if not one of the most important, one of the most interesting infectious diseases known. However, unlike brucellosis, Q fever does not constitute an economic problem for animal breeders, but only a problem for man's health."

The third chapter with the simple title "Rabies" is by Ernest S. Tierkel, Communicable Disease Center, Public Health Service, U.S. Department of Health, Education and Welfare, Atlanta, Georgia. The text covers 40 pages and is supported by just on 5 pages of references. A very brief historical introduction is followed by a succinct account of the clinical features of rabies in man and animals. Aetiology and pathology are briefly reviewed. In the section on epidemiology there is an impressive block graph showing the distribution of human deaths from rabies by age groups for the years 1944-54. The change in the position in subsequent years is shown by a study of morbidity trends. A detailed description of sylvatic rabies makes interesting and informative reading especially in regard to foxes, wolves, and bats. Prevention and diagnosis receive full attention.

Living attenuated virus vaccines for dogs prepared in chick embryos are held to be the most effective part of a rabies control programme. A significant advance in the prevention of human infection has been the development and use of hyper-immune rabies antiserum.

The fourth chapter, by R. M. du Toit of the Onderstepoort Veterinary Research Laboratories, Pretoria, Transvaal, South Africa, deals with the eradication of tsetse fly (*Glossina pallidipes*) from Zululand, Union of South Africa. Thirteen pages of text are followed by less than 1 page of references. Thermal smoke generators containing DDT was one method used; another was the application of BHC by means of aircraft. Dipping of cattle in DDT and bush clearance were supplementary methods. It is postulated that the successful conclusion of the eradication campaign in Zululand suggests that the methods could be successfully applied to other savanna species in Africa.

The fifth chapter on "Theilerioses" is contributed by W. O. Neitz, Division of Veterinary Services, Department of Agriculture, Onderstepoort, South Africa. Forty-nine pages of subject matter are supported by 8 pages of references. Each aspect of this important subject receives concise and informative treatment which makes this contribution a valuable source of information in a readily available form.

The sixth chapter breaks ground that may be new to readers in many parts of the world. By D. G. McKercher, School of Veterinary Medicine, University of California, the chapter describes infectious bovine rhinotracheitis, an acute contagious febrile infection of cattle, characterised by an intense inflammation of the upper respiratory passages and trachea. Apparently at present the disease is confined to continental United States though there are unconfirmed reports from Western Canada suggesting the possible presence of the disease.

The claim that a new disease has been recognised always arouses a certain amount of scepticism, but the writer contends that available information strongly suggests that infectious bovine rhinotracheitis is a new disease.

The final chapter in this volume deals with the important subject, "Tissue Culture in Veterinary Medical Research," and has been written by S. H. Madin, Naval Biological Laboratory, School of Public Health, University of California. Though covering 66 pages of text with 22 pages of references the chapter is essentially an informative introduction to this subject and cannot be expected, within its compass, to be more than that.

As is usual in these volumes there is provided an author index relating to the references given in each chapter and an adequate subject index.

The scope of the volume and the quality of its contents are such that the volume will be found to be an essential purchase by veterinary libraries. On the other hand, the diversity of its contents may make it difficult for the individual veterinary surgeon to decide on purchase of this particular volume of the series.

BOOKS RECEIVED

Protein and Amino-acid Nutrition, edited by Anthony A. Albanese (1959). Academic Press, New York and London. $16.00.

Physiology of Lactation (Revised fourth edition of Secretion of Milk by Espe and Smith), by Vearl R. Smith (1960). Fifth edition. Constable and Company Ltd., London. 45s.

Broilers (Production and Management), by Eric F. Golden and chapters by Keith Wilson, N.D.P., and Dr. J. L. Williams, D.S.O., M.B.E., M.I.BIOL. (1960). Second edition. Poultry World, London. 10s. 6d.

Development of the Poultry Industry in England and Wales, 1945-1959, by Rupert Coles, B.A., M.SC. (ECON.), M.SC. (AGRIC.), PH.D. (1960). Poultry World Ltd., London. 7s. 6d.

Collected Papers (Summary and Subject Reviews), Volume XVI. The Rowett Research Institute (Reid Library), Aberdeen.

Bibliographie des Deutschprachigen Schriftums zur Erforschung und Bekämpfung von Anzeigepflichtigen Tierseuchen, by Dorethee Rühlmann (1959). S. Hirzel Verlag, Leipzig C.1. Schuhmanchergässchen 1-3, Germany. DM. 20.60.

The Farmer's Dog, by John Holmes (1960). Popular Dogs Publishing Co. Ltd., London. 21s.

Explotación del Pato de Puesta, by José Antonio Romagosa Vilá (1957). Avigan, Apartado 726, Valencia, Spain. 150 Ptas.

Winter Keep on the Farm, by Ian Moore, M.SC., PH.D., N.D.A., DIP.AGRIC. (1960). Farmer and Stockbreeder Publications Ltd., London. 9s. 6d.

Aids to Forensic Pharmacy, by H. W. Fowler (1960). Baillière, Tindall & Cox Ltd., London. 12s. 6d.

Leitfaden der Ziegenkranheiten, by Prof. Med. Vet.

(*Concluded at foot of col. 1, page 371*)

News and Comment

INTENSIVE REARING OF CALVES

At the meeting of Council last month in Edinburgh the question was raised by one B.V.A. Division, through the Farm Livestock Committee, of the desirability of the present system of intensive rearing of calves for veal. The Technical Development Committee was asked to study the whole system of calf-rearing from the humanitarian and disease control aspects.

It would be of very great assistance if any members with first-hand knowledge of intensive rearing would send their observations to the Chairman of the T.D.C., at B.V.A. headquarters, 7 Mansfield Street, London, W.1. as soon as possible.

M.M.B. CATTLE BREEDING POLICY

At a Press Conference on April 28th, the Chairman of the Milk Marketing Board, Mr. Richard Trehane, outlined the Board's cattle breeding policy. He said that in recent weeks there had been some adverse criticism, mainly from the Friesian Society and from the National Cattle Breeders' Association, which had been fully discussed in the lay and farming press. The Board had refrained from making any statement at an earlier date because it was most anxious to discuss the matter with the two bodies concerned. Accordingly, Mr. Trehane himself had attended the council meeting of the British Friesian Society and he believed his explanations had done much to clear away some of the misconceptions which had grown up with regard to the Board's policy.

Mr. Trehane pointed out that when the Board opened its first cattle breeding centre 16 years ago it had envisaged an artificial insemination service for commercial cattle. As A.I. had developed, however, and the quality of the stud had been improved, pedigree breeders had been attracted to the service. The Chairman emphasised the Board's willingness to discuss the matter with all interested parties and to co-operate with all breed societies.

He explained in detail how panels, made up of a number of breeders and members serving on the Councils of the various breed societies, were responsible for the selection of bulls at each individual A.I. Centre. The selection of breeding stock was, in fact, a perfect example of teamwork between those who prepared and assessed the pedigrees of the various animals on the one hand, and those who inspected and assessed the qualities of the live animal. It was on the recommendation of the panels that the decision to use bulls extensively or to remove them from the stud rested in the final instance.

The present plan, which was being tried out in the northern breeding project, was an attempt to move towards concentrating on the use of bulls whose progeny had stood up to a critical assessment on conformation and who had revealed a satisfactory level of milk and butterfat production.

The statement to the press concluded:—

"The Board's breeding policy is not something new or revolutionary; its proposals are the logical and gradual development of principles which everyone has agreed are the best. Unfortunately the present controversy has aroused fears of in-breeding, and this has been dramatised by reference to the genetic proposition that 2,000,000 cows could be bred from 200 bulls without genetic deterioration. In anticipating fears of in-breeding the Board postulated the most extreme case of concentration of use that could be envisaged. This concentration is not within sight.

"We are satisfied, in the light of opinions expressed by leading authorities and because of the constant watchfulness of breeders serving on panels and the fact that we are drawing from a larger number of bulls and herds, that fears expressed on the dangers of in-breeding and loss of bloodlines are groundless. And we are not concentrating on breeding for milk alone—health, vigour, size, and temperament are just a few examples of important and practical aspects on which a careful watch is kept. In the selection of our sires care is taken to provide a broad base and to avoid undue concentration of a particular bull. A.I. provides special opportunity for safeguards of this kind to a degree hitherto not possible.

"The Board are determined to meet their responsibilities to the many users of the A.I. service, irrespective of the type or breed of cattle which they keep. That these efforts so far have been successful is shown by the fact that from a start in 1944 we now have 145,000 members and are breeding 1,500,000 cattle every year.

"We are prepared to work with all interested parties and if breed societies are fearful of competition it can in no sense be due to any desire of the Board to dominate the breeding of cattle. It may be that the high standard of bulls at A.I. centres will provide competition for private breeders, but at the same time this gives a new opportunity to them to examine to the full the results of their breeding. This is particularly true of the opportunity to progeny test in commercial conditions and on a scale not previously possible."

EUROPEAN AGRICULTURAL CONFERENCE

Organised by Messrs. Pfizer, this first conference was held in Lucerne from April 27th to May 1st. Over 160 delegates accepted invitations, including representatives of Austria, Belgium, Great Britain, Czecho-Slovakia, Denmark, Eire, Finland, France, Germany, Hungary, Italy, Netherlands, Norway,

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Books Received.—*Concluded.*

Wilhelm Schulze (1960). S. Hirzel Verlag, Leipzig C.1, Schumachergässchen 1-3, Germany. DM. 9.90.

Veterinary Applied Pharmacology and Therapeutics, by P. W. Daykin, B.SC., M.R.C.V.S., F.R.S.H. (1960). Baillière, Tindall & Cox Ltd., London. 55s.

British Small Animal Veterinary Association (Congress Proceedings, 1959), edited by Bruce V. Jones, M.R.C.V.S. (1960). Pergamon Press Ltd., Oxford. 55s.

Poland, Sweden, Switzerland, South Africa, the U.S.A., and Yugoslavia.

The general theme of the conference was "present and future aids to agricultural economy and livestock production"; and the programme was so designed as to provide symposia in each session followed by both controversial and confirmative data. The opening session was of a general nature. Suggestions were made as to how national research could be implemented internationally by such means as duplicate experimentation and trials carried out after forward planning; and how collaboration between research workers in nutrition and in veterinary medicine could be made closer. Other sessions dealt with such subjects as a possibly improved form of vitamin A, the use of so-called tranquillisers alone or with adjuncts such as stilboestrol in fattening bulls and cattle, together with growth studies in lambs, sheep and cattle. The medium-spectrum antibiotic "Oleandomycin" was discussed in relation to international research data on its use alone in broilers or in conjunction with other antibiotics such as terramycin and penicillin.

Modern type calf milk replacers, antibiotics in cattle and sheep fattening, modern creep feed starter rations for baby pigs, and antibiotics for growing pigs, poultry and calves were discussed and debated also. The final session provided several papers dealing with the importance of selected amino-acids and intact protein in the rations of growing pigs, and the possible extension of this approach to ruminants.

The party from Britain included the following members of the profession: Messrs. J. A. Anderson and W. E. Beattie, Dr. C. A. E. Briggs, Dr. J. J. Bullen, Messrs. J. K. Dow and W. Gibson, Dr. W. S. Gordon, Dr. W. R. Kerr, Messrs. R. Menard and K. H. B. Wood, and Dr. W. R. Wooldridge.

OPEN DAY AT EXPERIMENTAL FARM

A number of veterinary surgeons accepted an invitation to join a group of agricultural scientists and farmers who were guests at the British Oil & Cake Mills' Open Day last week on their experimental farm at Barlby, nr. Selby, Yorkshire.

Unfortunately, due to the risk of fowl pest, visitors were unable to see the poultry units, but the current experiments among pigs, and more especially among cattle, proved adequate to interest and stimulate all those who attended.

Work in the cattle unit follows 3 main lines:—

Firstly, progeny testing. B.O.C.M. are using the Danish method of progeny testing where groups of heifers sired by a number of bulls are compared for various characteristics, including production, under near-identical conditions. This method of progeny testing does not supplant the method used by the M.M.B. with their contemporary comparisons but is complementary to this system and, as is well known, has been adopted in Denmark for the improvement of dairy cattle. The characteristics looked for in the heifers include yield and quality of milk, plus general suitability for the dairy herd as judged by a panel of independent judges.

The second aspect of the cattle enterprise was the comparison of various breeds and cross breeds for beef production using early weaning methods and standard diets. It is of interest to note that apparently the best steers were of the South Devon breed and may forecast the use of South Devon bulls in crossing with dairy cattle.

Lastly, comparisons have been carried out on young Shorthorn beef bulls reared from calfhood at Barlby, and it was of some interest to note that the judgment of a prominent breeder closely approximated the results obtained by direct experimental observation.

B.O.C.M. should be congratulated on its development of the Barlby Experimental Farm which undoubtedly supplies a need in the cattle breeding industry. It is to be hoped that such privately sponsored progeny testing stations will be developed in other parts of the country, for undoubtedly they introduce new ideas and new thought into modern stock husbandry. It is understood that visitors are always welcome at Barlby, and for any veterinary surgeon interested in progressive stock breeding, such a visit would be amply repaid.

FAO CONFERENCE ON VETERINARY EDUCATION

As reported last week, this Conference was held in Church House, Westminster, from April 25th to 30th. A draft Report and draft Recommendation were received this week, but the subject matter seemed to be of too much importance for a hurried assessment. A full account, therefore, including a note on the social events, will be published in THE VETERINARY RECORD on May 14th.

R.C.V.S. OBITUARY

We record with regret the death of the following member of the profession:—

HEATHCOTE, Frederick Cyril, 3 Honley Road, Catford, London, S.E.6, Capt. late Royal Pioneers. Graduated December 20th, 1928, at the Royal Veterinary College, London. Died April 30th, 1960, aged 68 years.

R.V.C. REGISTRAR TO RETIRE

A correspondent writes: Past students of the Royal Veterinary College will learn with regret of the resignation of Mrs. K. Mitchell from the post of Registrar at the College.

She has been the Registrar for the past 20 years, and past students will remember the active interest she has always taken in the affairs of her students both in and out of College. She will be greatly missed.

The present students of the R.V.C. have decided to make a presentation to Mrs. Mitchell, and it is felt that there may well be past members of the College who wish to be associated with it.

In order that they may do so they are invited to send their subscriptions to the Secretary, Students Union, R.V.C. Camden Town, N.W.1, before May 28th. Cheques and postal orders should be made out to: Students Union, Mrs. Mitchell Testimonial Fund.

PERSONAL

Mr. D. Cameron, Deputy Regional Veterinary Officer, remains at Norwich.

Mr. R. J. Garner, of the A.R.C. Field Station, Compton, has been appointed Head of the Public Health Section, Authority Health and Safety Branch Radiological Protection Division, U.K.A.E.A., Harwell.

Mr. J. Simpson took up appointment as Deputy Regional Veterinary Officer at Head Office, Tolworth, on May 2nd.

Mr. J. Small, Divisional Veterinary Officer, Forfar, and **Mr. A. J. Beeson,** Divisional Veterinary Officer, Maidstone, retired from the Service as from April 30th and May 3rd respectively.

Mr. G. Tullis took up appointment as Deputy Regional Veterinary Officer at Newcastle upon Tyne on May 2nd, 1960.

Engagements

ANDERSON—KIÆR.—The engagement is announced between Ronald Shand Anderson, B.V.M.S., M.R.C.V.S., only son of Mr. and Mrs. J. M. Anderson, 34, Lenzie Road, Stepps, Glasgow, and Pauline Kiær, eldest daughter of the late Mr. H. T. Kiær and of Mrs. Kiær, Oslo.

WESTAWAY—REEVE.—The engagement is announced between Arthur John Westaway, B.V.SC., M.R.C.V.S., of Minstead, Keydell Avenue, Horndean, Hants., and Eileen Lilian Reeve of Leighton Buzzard, Beds.

Marriages

STOCK—SCANTLEBURY.—At St. Peter's, Staines, on April 23rd, 1960, the marriage took place between John Edward Stock, B.VET.MED., M.R.C.V.S., and Anne Petrea Scantlebury, A.G.S.M., L.R.A.M.

WALTER—WARD.—At Epsom, Surrey, on March 19th, 1960, Andrew J. Walter, B.SC. (VET. SCI.), M.R.C.V.S., to Margaret V. Ward, M.B., B.S., M.R.C.S., L.R.C.P.

Births

DOVEY.—On April 19th, 1960, at St. Joseph's Hospital, Port Elizabeth, South Africa, to Daphne, wife of John Dovey, D.F.M., M.R.C.V.S., a fifth daughter, Heather Barbara.

HUMPHREYS.—On April 20th, 1960, to Susan, wife of Medwyn Humphreys, M.R.C.V.S., of "Eryl," Vale Street, Denbigh, a daughter, Carolyn Ann, sister to Martin and Bruce.

MARTIN.—On April 22nd, 1960, at Nuneaton, Warwicks., to Margaret and Brian Martin, a daughter.

MURGATROYD.—On April 22nd, 1960, to Josephine (*née* Briggs, B.SC.) and Alfred E. Murgatroyd, B.V.SC., M.R.C.V.S., of 61, Knutsford Road, Warrington, a son, Damon.

COMING EVENTS

May

11th (Wed.). Annual General Meeting of the B.S.A.V.A. Midland Area at the Queen's Hotel, Birmingham, 8 p.m.

12th (Thur.). Meeting of the South-Eastern Veterinary Association at the Royal Star Hotel, Maidstone, 7 p.m.

Annual General Meeting of the Herts. and Beds. Veterinary Society at Hawkshead House, Hawkshead Lane, North Mimms, Hatfield, 2.30 p.m. The Summer Outing will follow immediately after the meeting.

13th (Fri.). Meeting of the Welsh Branch at the Belle Vue Hotel, Aberystwyth, 2.15 p.m.

18th (Wed.). Meeting of the Lancashire Veterinary Association at the Queen Hotel, Chester, 7.30 p.m.

Annual Meeting of the Supplementary Veterinary Register Division at 7, Mansfield Street, Portland Place, London, W.1, 2.30 p.m.

Annual General Meeting of the Society of Practising Veterinary Surgeons in the Lecture Theatre, School of Veterinary Medicine, Madingley Road, Cambridge, 2.30 p.m.

19th (Thurs.). Annual General Meeting of the Dumfries and Galloway Division in the Station Hotel, Dumfries, 2 p.m.

20th (Fri.). Annual General Meeting of the Midland Counties Veterinary Association at Avoncroft Cattle Breeders Ltd., Sugarbrook.

Meeting of the B.S.A.V.A. South Eastern Region at the Royal Mount Ephraim Hotel, Tunbridge Wells, 8 p.m.

21st (Sat.). Annual Reunion and Dinner Dance of the Chiron Club at the Grand Atlantic Hotel, Weston-super-Mare, 7 p.m.

22nd (Sun.). R.V.C. Annual Motor Rally to Beachy Head.

25th (Wed.). R.C.V.S. Day.

117th Annual General Meeting of the Royal College of Veterinary Surgeons at 10, Red Lion Square, London, W.C.1, 3.45 p.m.

Annual General Meeting of the Sussex Veterinary Society at the Old Ship Hotel, Brighton, 2.30 p.m. Annual General Meeting of the Society of Women Veterinary Surgeons at Imperial Chemical House, Millbank, London, S.W.1, 2.15 p.m.

Annual General Meeting of the Victoria Veterinary Benevolent Fund at 10, Red Lion Square, London, W.C.1, 4.15 p.m. (or immediately following the Annual General Meeting of the R.C.V.S.).

26th (Thurs.). Meeting of the Western Counties Veterinary Association at the Hotel Victoria, Newquay, 2.30 p.m.

27th (Fri.). Dance of the Herts. and Beds. Veterinary Association Ladies' Guild at the Dujon, Bedford, 8.30 p.m.

30th (Mon.). Meeting of the East Yorkshire Clinical Club at the Talbot Hotel, Malton

June

3rd (Fri.). 168th Annual Ball of the R.V.C. Students' Union Society at the Park Lane Hotel, Piccadilly, W.1, 8 p.m.

10th (Fri.). Annual Dinner of the R.A.V.C. Officer's Club at the Naval and Military Club, 94, Piccadilly, London, W.1.

July

6th (Wed.). Meeting of the Technical Development Committee of the B.V.A., at 7, Mansfield Street, 10.30 a.m.

20th, 21st and 22nd (Wed., Thurs. and Fri.). B.V.A. Committee and Council Meetings in London.

August 28th to September 3rd. 78th Annual Congress of the British Veterinary Association in Glasgow.

NEW LABORATORIES OPENED

The Minister of Health opened the new building and laboratories of Messrs. Smith, Kline & French at Welwyn Garden City on April 28th. Parties of visiting doctors and veterinary surgeons were taken on a tour of the laboratories and plant and this tour included a demonstration of the coloured television unit. It will be used, by courtesy of the Company, at the B.V.A. Annual Congress at Glasgow. Among members of the profession present were Mr. S. L. Hignett, President of the B.V.A., Dr. R. F. Gordon, Professor L. P. Pugh, Dr. W. R. Wooldridge, Mr. L. G. Anderson, and Mr. H. I. Field.

ADDRESSES OF DISEASE INFECTED PREMISES

The list given below indicates, first the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak.

Anthrax

Aberdeen. Foreside, Premnay, Insch (Apr. 25).

Gloucs. The Next, School Lane, Longhope (Apr. 25).

Kirkcudbright. Balannan, Ringford, Castle Douglas (Apr. 28).

Salop. Bank Farm, Gatten, Pontesbury, Shrewsbury (Apr. 25); Pitchford Park Farm, Pitchford, Condover, Shrewsbury; Victoria House, Worthen, Shrewsbury (May 2).

Yorks. Village Farm, Laverton, Kirkby Malzeard, Ripon (Apr. 27).

Fowl Pest

Beds. The Priory, Stanbridge, Leighton Buzzard; No. 5, Molevers Lane, Bromham (Apr. 22); Manor Farm, Sewell, Dunstable (Apr. 23); Elsdene, Church Lane, Little Gransden, Sandy; Springfield, Wellhead, Dunstable; Good Intent Farm, Edlesborough, Dunstable (Apr. 25); 71, School Lane, Eaton Bray, Dunstable (Apr. 26); Smallholding, Cave Lane, Allotments, Bedford (Apr. 27).

Bucks. Hundreds Farm, Woburn, Bletchley (Apr. 28).

Cambs. Mill View, Caxton (Apr. 22); 1, Rosemary Lane, The Americas, Sutton, Ely (Apr. 28); 49, The Row, Sutton, Ely (May 2).

Essex. Thistley Hall, Widdington, Saffron Walden (Apr. 29); The Warren, Wendon Lofts, Saffron Walden (Apr. 30); Lippingwells Farm, Rushley Green, Castle Hedingham, Halstead (May 2).

Hants. Dunster Cottage, The Frenches, E. Wellow, Romsey; Railston-Brown, Dublin Farm House, Wherwell, Andover (Apr. 23).

Herts. The Hoo Poultry Farm, Whitwell, Hitchin (Apr. 30).

Hunts. Woodlands, Ellington Thorpe, Ellington (May 2).

Leicester. 24, King Street, Whetstone (Apr. 27).

Lincs. The Poplars, Collyweston, Stamford; Greystones, Collyweston, Stamford (Apr. 22); Blacksmith, Main Road, Collyweston, Stamford; 3, Main Road, Collyweston, Stamford; 7, Kingscliffe Road, Collyweston, Stamford; Back Lane, Great Ponton, Grantham (Apr. 26); Collyweston Road, Collyweston, Stamford (Apr. 28); 7, Whitham Town, Boston; 2, Coronation Villas, Tinwell, Stamford (Apr. 29).

Notts. Archway House, Kirklington, Newark (Apr. 24); Chestnut Farm, Normanton, Southwell; Gables Farm, Bilsthorpe, Newark; Home Farm, Bilsthorpe, Newark (Apr. 25); Orchard View, Blidworth Road, Calverton; Middle Corkhill Farm, Kirklington, Newark; 2, Railway Cottages, Bilsthorpe, Newark; Far Baulker Farm, Oxton, Newark; Corner Shop, Rolleston, Newark (Apr. 26); Poultry Farm, Rolleston, Newark; Halam House Farm, Halam, Newark (Apr. 27); Lockwell Hill Farm, Farnsfield, Newark; Diamond Cottage, Greaves Lane, Edingley, Newark; Manor Farm, Normanton, Southwell (Apr. 28); Lower Kirklington Road, Southwell, Burtons Buildings, Baxter Lane Sibthorpe, Newark; Farnsfield Laying Site, Longlands Lane, Farnsfield, Newark; Little Corkhill Farm, Kirklington, Newark; Sunnybank Farm, Elston Road, East Stoke; Newark Collery Tip Group, Ollerton, Newark; Robin Hood Farm, Bilsthorpe, Newark (Apr. 29); Ollerton Hills Site, Ollerton, Newark (Apr. 30); Rufford Park, Rufford, Ollerton, Newark; The Grapes Inn, Southwell; Crookford Piggeries, Elkesby, Retford; Innisdoone, Peafield Lane, Mansfield Woodhouse; 151, Clipson Road West, Forest Town, Mansfield (May 1).

Suffolk. Ryes Farm, Gosbeck, Ipswich (Apr. 30).

Warwicks. 33, Needlers End Lane, Balsall Common, Coventry (Apr. 22); 58, Beacon Hill, Rubery, Rednal, Birmingham (Apr. 23); Brickhill Mink Farm, Coleshill (Apr. 24); Rubery Hill Hospital, Birmingham (Apr. 25); Green Farm, Shustoke, Coleshill, Birmingham (Apr. 26); Croft Farm, Shustoke, Coleshill, Birmingham (Apr. 27); Newnham Lodge Farm, Monks Kirby, Rugby (Apr. 29); Green Lodge, Shustoke, Coldhill, Birmingham (May 1).

Worcs. Impney Farm, Droitwich (Apr. 24); Berry Hill Farm, Droitwich; 5, Morton Road, Fernhill Heath (Apr. 30); 35, Wildmoor Lane, Catshill, Bromsgrove (May 2).

Yorks. Whitegate Farm, Oakworth, Keighley (May 2).

Swine Fever

Ches. Walkers Green Farm, Sproston, Middlewich (May 2).

Cornwall. Trewindle Farm, Lostwithiel (May 2).

Devon. Broadmoor, Beaworthy (Apr. 26); Suddon Farm, Newton St. Petrock, Torrington; Hartgrove Farm, Musbury, Axminster.

Essex. Fairlands Farm, Abbess Roding, Ongar; Newlands Farm, Cranfield Park Road, Nevendon, Wickford (Apr. 28).

Hants. Veyers Farm, Gatcombe, Newport, Isle of Wight (Apr. 25); Chale Abbey Farm, Chale, Ventnor, Isle of Wight (Apr. 29); Beaulieu Cottage, Appuldurcombe Road, Wroxall, Isle of Wight (May 2).

Herts. The Maltings, Sheering Lower Road, Sheering, Bishops Stortford (Apr. 26).

Kent. Oakwood Hospital Farm, Maidstone (Apr. 28).

Lancs. Park Farm, Warburton, Lymm, Warrington (Apr. 22).

Lincs. The Bye Pass, Main Road, Wrangle, Boston (Apr. 26).

Middlesex. St. Leonards Farm, New Years Green, Harefield, Uxbridge (Apr. 27).

Notts. Piggeries, Nether Headon Camp, Retford (Apr. 22); Lodge Farm, Westborough, Newark (Apr. 29).

Somerset. No. 10, Lockleaze Holdings, Horfield, Bristol, 7 (Apr. 25); Foxcombe Farm, Galhampton, Yeovil (Apr. 28); Hurmans Farm, Woolavington, Bridgwater (May 2).

Staffs. The Firs, Levedale Road, Longridge, Dunston (Apr. 22); Baldwins Gate Farm, Baldwins Gate, Newcastle (Apr. 25); Bignall End Farm, Bignall End, Stoke-on-Trent (Apr. 26).

Suffolk. High View, Yarmouth Road, Corton, Lowestoft (Apr. 26); Hall Farm, Whepstead, Bury St. Edmunds (Apr. 28); Top Road, Ilketshall St. Andrews, Beccles (Apr. 29); New Road, Hurdle Drove, West Row, Bury St. Edmunds (May 2).

Surrey. Broadwater Farm, Weybridge (May 2).

Sussex. Eighteen Pounder Farm, Three Oakes, Guestling, Hastings (Apr. 29).

Warwicks. Holt Far, Bruntisthorpe, Rugby (Apr. 25).

Wilts. Home Farm, Spye Park, Bromham, Chippenham (Apr. 28).

Letters to the Editor

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the B.V.A.

SCIENTIFIC

Otodectic Mange in the Dog

Sir,—An additional 100 unselected and untreated cases of otitis externa in the dog have been examined for the presence of *Otodectes cynotis*, and the findings are very similar to those previously reported (*Vet. Rec.* **70.** 740-2). The purpose of this letter is again to draw attention to the fact that *Otodectes* is an important cause of otitis externa in the dog. Mites were found in 46 dogs, of which 43 had bilateral and 3 unilateral infections; 2 of the unilateral cases had a suppurative infection of the other ear. Auriscopical and parasitological examinations were positive in 29 dogs; the clinical examination alone revealed a further 10 cases and the laboratory method 7 additional infections. Of the 100 dogs examined, 58 were males, and 42 were bitches; their ages ranged from 6 weeks to 13 years. There was no breed predilection but, as found in the first survey, there was an inexplicable higher incidence in males as approximately 53 per cent. harboured mites, whereas approximately 35 per cent. only of the bitches were affected.

In the period November to April about 59 per cent. of cases were positive, whereas from May to October 37 per cent. were positive. Nine dogs had a profusion of mites in the ears but the remaining 37 showed fewer than 3 detectable mites on auriscopic examination. The possible source of contagion was investigated as closely as possible, and in 26 dogs history and age suggested the dam and/or the breeding establishment. Nine of these 26 animals lived with cats, of which 3 harboured mites, 2 did not, and 4 were not presented for examination. Another 2 of this group of dogs could have become infected in kennels, one was in contact with an infected dog and one with an unexamined dog. Fifteen infected mature dogs lived with cats, of which 3 harboured mites and 6 were not presented for examination. The remaining cat had a bilateral suppurative otitis and had lived for 7 years with the affected dog which had a very long history of otitis and was not presented for examination until an ulcerated ear tip had developed. Two infected dogs had a history directly related to a stay in kennels. One dog was in contact with a proven affected dog, and another was in contact with an unexamined dog. There was one case in which the history did not indicate the source of contagion.

Yours faithfully,
R. CHURCHILL FROST,
W. P. BERESFORD-JONES.

Royal Veterinary College,
Royal College Street,
London, N.W.1.
April 20th, 1960.

GENERAL

The Well of English

Sir,—I was pleased to read Dr. E. L. Taylor's letter in your edition of April 30th because, as a lecturer in Agricultural Zoology, I find the problem of a common name for *Bos taurus* a very real one. In the quotation from the O.E.D. "commonly" should not be confused with "correctly" because the use of "ox" is surely right in this connection. However, I would agree that the name does not come easily to the tongue and nowadays may confuse an audience. Would not an easy answer be to use the term "cattle" both as singular and plural as in "sheep"?

Yours faithfully,
S. J. WILLIS.

Shuttleworth College,
Old Warden Park,
Biggleswade,
Bedfordshire.
May 3rd, 1960.

Sir,—Your recent Editorial reminded me of a case report which appeared in THE RECORD some time ago. I would not wish to disparage the article from a clinical point of view, but it left me wondering whether I had picked up the *Lancet* by mistake. Describing acute septicaemia in a Thoroughbred, the author wrote: "The stable headman informed me that he had not finished his night feed and had a fever. The only thing the groom could tell me was that he was inclined to lie down more than usual in the past few days. As I approached the stable, he was walked out towards me and appeared to stagger. I had him placed in a large rolling ring some 20 yards away."

Another correspondent once remarked that Major Hamilton Kirk "still has a kick to defend any act of cruelty to an animal," but I feel certain that he did not really mean what he said.

Yours faithfully,
A. L. BONGARD.

5, Ann Street,
Enniskillen,
Co. Fermanagh.
May 2nd, 1960.

An Anaesthetic Mishap

Sir,—The following unfortunate sequel to an anaesthetic mishap in a cat may be of interest. To the best of my belief, whilst there is no dearth of literature concerning effects of cerebral anoxia in experimental animals, little, if anything, has been recorded of recent years in clinical cases.

The patient was a young adult female cat undergoing ovaro-hysterectomy. Anaesthesia was by the

method I routinely use in this practice for short operations on cats, oxygen bubbled through trichloroethylene (Trilene) *via* a Hall's face mask. About midway through operation, *i.e.* after some 2 minutes' surgical anaesthesia, respiratory failure occurred followed in some 30 seconds by cardiac arrest.

Resuscitation was by cardiac massage (*via* the intact chest wall), artificial respiration and finally intracardiac injection of adrenalin. These methods were successful in restarting heart action and subsequently respirations, and in about 10 minutes the cat was breathing regularly and deeply and cardiac rhythm was virtually normal.

In view of the urgency of the situation no time-keeping was thought of but all 3 persons present are agreed that cardiac arrest and thus cerebral anoxia lasted about 5 minutes.

The cat made a slow recovery from anaesthesia and had a period of apparent narcotic excitement some 3 hours or more after the accident.

When the cat was fully recovered from anaesthesia it became obvious that she was totally blind. She was kept for one week in the hope that sight might be regained. During this week she showed no interest in food but accepted protein hydrolysate perfectly co-operatively when hand fed. Allowing for the sudden and complete loss of sight her gait and co-ordination seemed normal. On the eighth post-operation day she was finally examined before euthanasia was undertaken. Pupillary reflexes were brisk and normal, both optic discs were normal as also were the blood vessels, bodily condition was fair; blindness was complete and so far as could be ascertained the cat was deaf; she showed no response when attractive and pungent food was held near her nose so we assumed the sense of smell to be lost too. I was thus faced with the disconcerting and unpalatable situation that, having apparently "saved" an anaesthetic casualty, I now had to destroy her on humane grounds owing to severe brain damage resulting from cerebral anoxia. So does pride go before a fall!

I was greatly concerned at the initial anaesthetic accident since in over 10 years' use of trichloroethylene as my routine gaseous anaesthetic for cats I had never once had respiratory failure, in fact in some 2,000 anaesthesias with this agent I think I am right in saying I have only lost 3 patients, the one recorded by me in 1950 (cardiac failure), one other cardiac failure during an unusually prolonged operation and the third cause unknown since she was removed from the operating table perfectly normal and was found dead 15 minutes later.

I subsequently found the cause of respiratory failure to be one of those human errors which ought not to occur but do sometimes happen to all of us, namely that the expiratory valve which should be well open when used with the tight-fitting Hall's mask and which I routinely check at the start of any anaesthetic session was found to be very tightly closed, a mystery which I can never solve.

This episode will make me very dubious in future of continuing resuscitation attempts beyond some 3 minutes after cardiac arrest.

I know many people must have successfully resuscitated patients following anaesthetic accidents and should be interested to know if any further cases of brain damage due to anoxia have occurred.

Yours faithfully,
JOAN O. JOSHUA.

78 Brentway,
Finchley, N.3.
April 26th, 1960.

Reference

JOSHUA, J. O. (1950). *Vet. Rec.* **62**. 191.

The Things Our Clients Say

Sir,—A man and his wife brought their dog in to me, in distress because he had been out in the nettles and his feet were driving him mad. I gave him the usual attention and a Largactil tablet to keep him quiet. In the course of the conversation, I mentioned that the tablets were also useful on Guy Fawkes night.

"Why," said the man, "do they go off bang?"

Yours faithfully,
V. P. T. TROUGHT.

99, High Street,
Rochester,
Kent.
April 26th, 1960.

DISEASES OF ANIMALS ACT, 1950—GREAT BRITAIN
Summary of Returns of Confirmed Outbreaks of Scheduled (Notifiable) Diseases

Period		Anthrax	Atrophic Rhinitis	Foot-and-Mouth	Fowl Pest	Sheep Scab	Swine Fever
16th to 31st March, 1960		12	—	—	57	—	53
Corresponding period in	1959	13	—	—	23	—	65
	1958	15	1	3	22	—	56
	1957	31	—	2	19	—	32
1st Jan. to 31st Mar., 1960		79	—	10	882	—	285
Corresponding period in	1959	63	—	1	2[illegible]8	—	288
	1958	52	2	100	195	—	268
	1957	133	2	[illegible]5	251	—	213